

## CASE REPORT

# Transient appearance of antegrade conduction via an AV accessory pathway caused by atrial fibrillation in a patient with intermittent Wolff-Parkinson-White syndrome

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## Abstract

**A 55 year old man with intermittent Wolff-Parkinson-White (WPW) syndrome had an episode of atrial fibrillation (AF) that lasted for 117 days. After interruption of the AF a  $\Delta$ wave appeared that lasted for two days and then disappeared. Exercise stress and isoprenaline infusion could not reproduce the  $\Delta$ wave, but after another episode of AF which lasted for seven days a persistent  $\Delta$ wave appeared that lasted for six hours. In an electrophysiological study performed on a day without a  $\Delta$ wave, neither antegrade nor retrograde conduction via an accessory pathway was seen, but after atrial burst pacing (at 250 ms cycle length) for 10 minutes, a  $\Delta$ wave appeared lasting for 16 seconds. Atrial electrical remodelling—that is, the shortening of the atrial effective refractory period caused by AF, is a possible mechanism of the appearance of the  $\Delta$ wave.**

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**Keywords:** Wolff-Parkinson-White syndrome; atrial fibrillation; electrical remodelling

Several mechanisms have been suggested to underlie the intermittent appearance of antegrade conduction via an atrioventricular accessory pathway in patients with intermittent Wolff-Parkinson-White (WPW) syndrome.<sup>1-4</sup> In some of these mechanisms, the refractoriness of the accessory pathway itself or the atrial muscle close to the atrial connection of the accessory pathway is thought to play an important role in the intermittency. Refractoriness is an intrinsic electrophysiological property of the cardiac muscle, but it can be modified by heart rate, the tone of the autonomic nervous system, serum electrolytes, hypoxia, cardioactive drugs, and other factors.<sup>1-4</sup> It has been reported that the continuation of tachyarrhythmia can also modify the refractoriness of the cardiac muscle, a phenomenon that is now understood as “electrical remodelling”.<sup>5-8</sup>

We present a patient with intermittent WPW syndrome, in whom persistent antegrade conduction via an accessory pathway—that is, a  $\Delta$ wave, appeared transiently only after the interruption of atrial fibrillation (AF).

## Case report

A 55 year old man was diagnosed with intermittent WPW syndrome in 1987 but the  $\Delta$ wave was rarely observed. When he visited a local hospital, the surface ECG showed sinus rhythm and incomplete right bundle branch block, but no  $\Delta$ wave was observed (fig 1A).

### FIRST EPISODE OF AF

When the patient visited his local hospital again because of palpitation on 29 March 1997, the surface ECG showed AF and no  $\Delta$ wave was observed at this time. He was prescribed oral procainamide (1000 mg/day) but the AF continued (fig 2). The  $\Delta$ wave was documented first in this episode on 19 April, but its appearance was intermittent and the shortest RR interval with the  $\Delta$ wave was 460 ms. Because of the continuation of AF, the patient developed congestive heart failure. Because of the negative inotropic action on cardiac muscle, procainamide was discontinued on 16 July. His heart failure improved during hospitalisation, but AF continued and the shortest RR interval with the  $\Delta$ wave was gradually shortened (fig 2). Catheter ablation of the accessory pathway was considered and the patient was moved to our hospital on 16 July 1997. At the time of admission the ECG showed AF and an intermittent appearance of the  $\Delta$ wave; the shortest RR interval with the  $\Delta$ wave was 320 ms (fig 1B). On 23 July, before a scheduled electrophysiological study, his cardiac rhythm went from AF to sinus rhythm. The AF had lasted for 117 days. Immediately after interruption of the AF the  $\Delta$ wave was persistently observed (fig 1C); however, it disappeared about two days after the interruption of AF (fig 1D). During this episode of the appearance and disappearance of the  $\Delta$ wave, no antiarrhythmic drugs were used (fig 2).

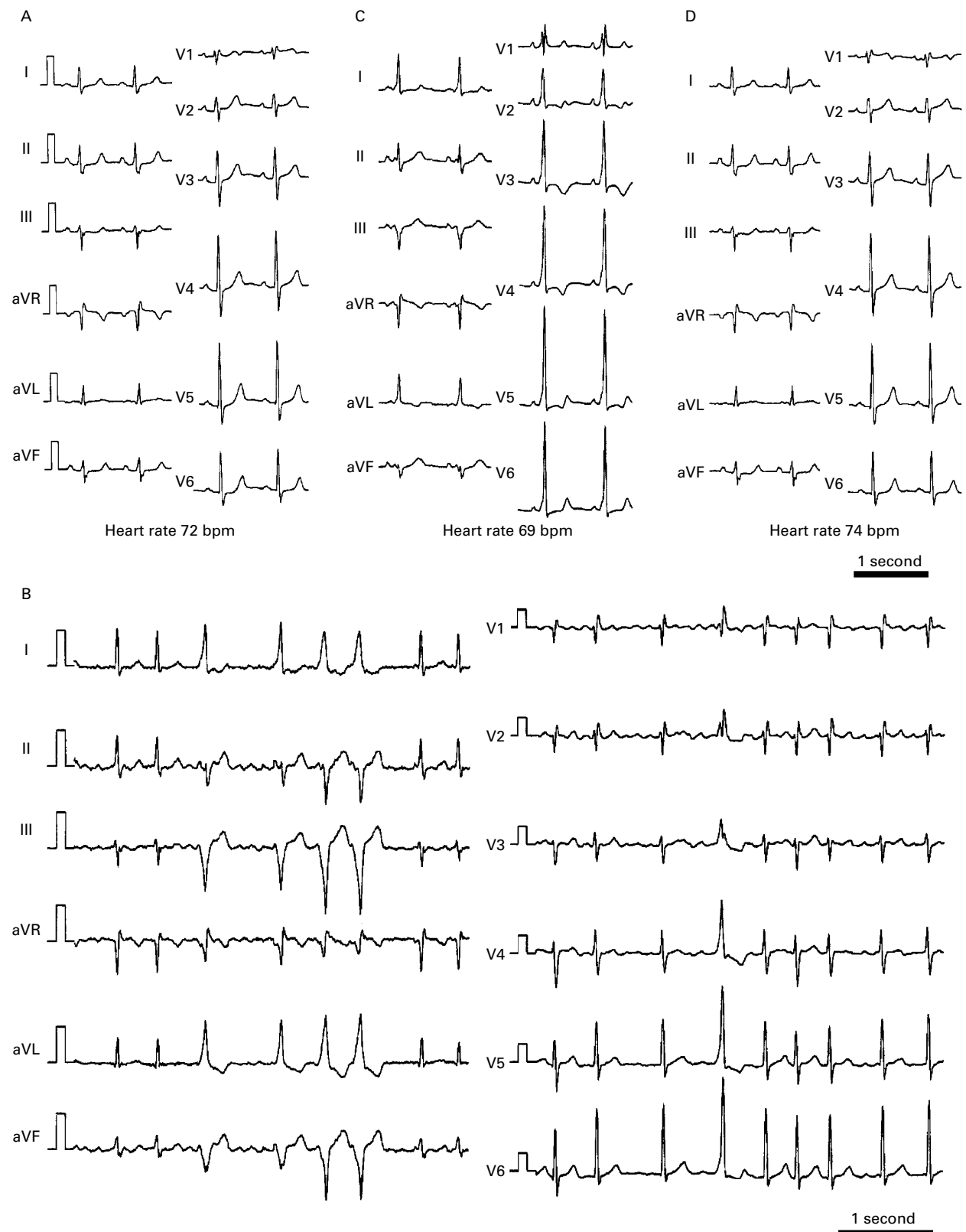
### HOLTER MONITORING

Holter monitoring was performed on a day with  $\Delta$ wave appearance and a day without  $\Delta$ wave appearance. There was no significant difference in autonomic tone between these two days.

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**Figure 1** 12 lead ECG recordings of the patient during his clinical course. (A) Before the first episode of AF (B) During AF, which was approximately 100 days after the onset of the first episode of AF (C) One day after the interruption of the first episode of AF (D) Four days after the interruption of the first episode of AF

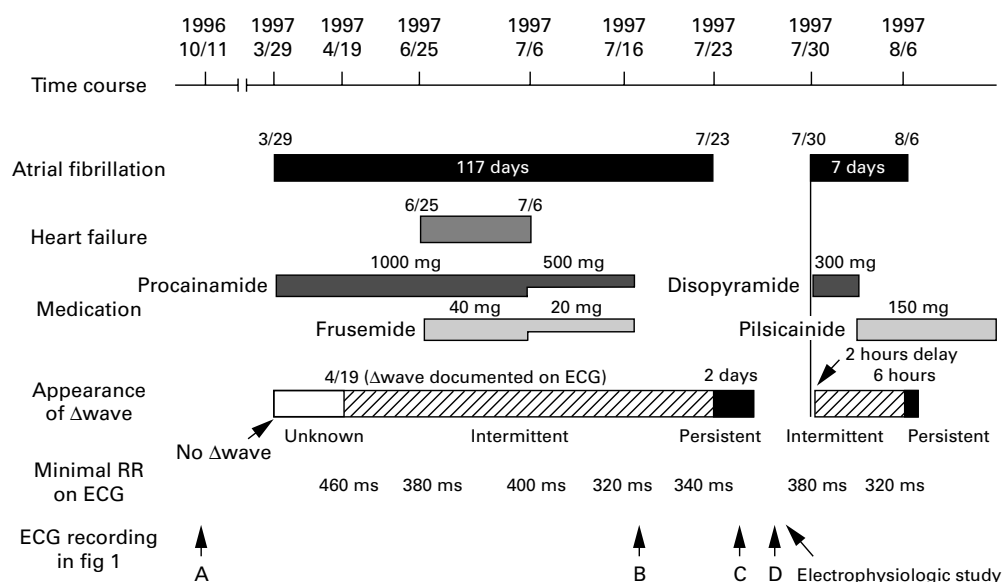


Figure 2 Clinical course of the two episodes of AF and transient appearance of the Δwave. Note that the persistent appearance of the Δwave was observed only transiently after the interruption of AF. There was some time delay between the onset of the AF and the time of the Δwave appearance, and the minimal RR interval with the Δwave shortened gradually during the episodes of AF.

#### EXERCISE STRESS TEST

A treadmill stress test was performed on a day without Δwave appearance. Heart rate increased from 62 to 142 beats/min during exercise, but no Δwave was observed in the recording throughout the study.

#### ELECTROPHYSIOLOGICAL STUDY

An electrophysiological study was performed on a day without Δwave appearance. The programmed electrical stimulation protocol, which included single extrastimuli after two basic drive train cycle lengths (400 and 600 ms) and transient pacing with fixed cycle lengths (ranging from 667 to 286 ms) was delivered from two right atrial sites, the coronary sinus and the right ventricular apex. The study did not document any conduction through the accessory pathway, which was suspected to be located in the left lateral posterior side free wall, based on the shapes of the Δwave in the surface 12 lead ECG (fig 1C). Isoprenaline was infused intravenously at a dose up to 1 mg/kg/hour and the whole pacing protocol was repeated, but no accessory pathway conduction was documented. The shortening of the refractoriness of the atrial muscle or the accessory pathway itself by atrial electrical remodelling<sup>6</sup> was considered as a possible cause of the accessory pathway conduction; therefore, atrial burst pacing for 10 minutes at a cycle length of 250 ms was performed at the low right atrial site. Immediately after the interruption of the atrial pacing, the Δwave appeared lasting for only 16 seconds. The effective refractory period measured at the right low lateral site was shortened from 202 to 188 ms.

#### SECOND EPISODE OF AF

On 30 July, AF recurred and lasted for seven days (fig 2). Intravenous disopyramide (50 mg) and oral disopyramide (300 mg/day) for three

days did not interrupt the AF, therefore, oral pilsicainide (150 mg/day) was administered, and the AF was interrupted on the third day. During this episode of AF, no Δwave was observed initially, but it started to appear about two hours after the onset of AF, and its appearance was intermittent. As in the first episode, the Δwave became persistent immediately after the interruption of the AF, but it lasted only about six hours this time. Oral pilsicainide was used for the interruption and prevention of AF during the second episode of appearance and disappearance of the Δwave. With the continuation of oral pilsicainide, AF did not recur for up to eight months.

#### Discussion

A simple phase 3 or 4 block is not the likely mechanism of the transient appearance of Δwaves in our patient because heart rate dependency was not observed.<sup>1</sup> Because the electrophysiological properties of the accessory pathway could not be evaluated in the electrophysiological study, the participation of the decremental property and the impedance mismatch is not known. Influence of the autonomic tone is unlikely as there was no significant difference in autonomic tone in the heart rate variability analysis between the days with and without Δwave appearance. Retrograde concealed conduction is also an unlikely mechanism as no pattern of electrical stimulation could document the conduction through the accessory pathway.<sup>3,4</sup> A change in atrial activation sequence is also an unlikely mechanism because pacing at various atrial sites could not document conduction through the accessory pathway during electrophysiological study. Because (1) the Δwave appeared transiently only after interruption of AF; (2) the minimal RR interval with the Δwave shortened gradually during the episodes of AF; and (3) atrial burst pacing reproduced the Δwave

appearance, and the atrial effective refractory period was shortened in the electrophysiological study, the shortening of the refractoriness of the atrial muscle or the accessory pathway itself—that is, electrical remodelling, is thought to be at least part of the mechanism of this phenomenon in our patient.<sup>5–8</sup> The role of antiarrhythmic drugs in transient appearance of the  $\Delta$ wave is unclear; however, the action of class Ia drugs does not seem to be a determining factor because the  $\Delta$ wave appeared after discontinuation of procainamide in the first episode but during continuation of pilsicainide in the second episode.

It has been documented that continuous rapid atrial excitement causes a shortening of the atrial refractoriness as well as a decrease in its rate dependency—that is, atrial electrical remodelling.<sup>5,6</sup> It is unclear whether similar electrical remodelling can occur in conduction through atrioventricular accessory pathways. In patients with manifest WPW syndrome, rapid atrial excitation such as AF causes a rapid ventricular response, especially in patients with short refractoriness of the accessory pathway—that is, pseudo-ventricular tachycardia, so that it is practically impossible to follow up such patients for a longer period in AF rhythm.<sup>1</sup> Accessory pathways are thought to originate anatomically from atrial tissue, and therefore it seems possible for an accessory pathway itself to show electrical remodelling, similar to atrial

tissue.<sup>1</sup> In a patient with intermittent WPW syndrome, the anatomical level of the block of the conduction through the accessory pathway is unclear. It could be in the accessory pathway itself or in the atrial or ventricular muscles close to the accessory pathway connection. Although we hypothesised that electrical remodelling was a mechanism for the transient appearance of the  $\Delta$ wave in our patient, the site that showed shortening of the refractoriness by atrial remodelling could not be determined.

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